pp. 293—355.] [January 30, 1913.

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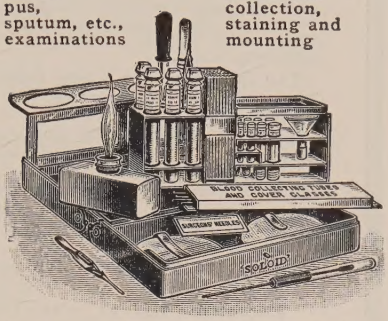

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TROPICAL DISEASES BUREAU.

TROPICAL DISEASES BULLETIN.

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PELLAGRA.

ROBERTS (Stewart R.). **Pellagra: History, Distribution, Diagnosis, Prognosis, Treatment, Etiology.** 272 pp. With 89 special engravings and a coloured frontispiece. 1912. London: Henry Kimpton. [12*s*. net.]

This is a useful well-illustrated book written for the student and the practising physician. The author tells us that pellagra is not contagious, a necessary statement in Georgia where he says he has known unfortunate patients shunned by their timorous relatives. Like other writers he agrees that it is not inherited though it may be a cause of race degeneracy.

It is estimated that there are now 2,500 cases of pellagra in Georgia, or one fourth of the total number of individuals attacked in the United States. Professor Roberts gives among the types of the disease an acute or fulminating form which lasts from one week to three months and progresses rapidly to death. He proposes to substitute for STRAMBIO'S term, pellagra *sine* pellagra, or pellagrous disease without skin lesions, "pellagra *sine* exanthemata" as being less misleading. [But is it necessary to coin the hybrid "dermotagra" for rough skin?]

The chapter on the changes in the nervous system, which is the longest in the book, is enriched by many excellent photographs contributed by Dr. E. BRAVETTA of the Mombello asylum, near Milan. Stress is laid upon the undoubted fact that pellagra in the United States, as compared with the disease in Europe and Egypt, is more severe, more rapidly progressive and proportionately more fatal.

In discussing prognosis the author reminds us that recovery from the skin lesions does not mean that the patient is cured, that a gradual increase of weight is the best sign of general improvement, and that surgeons should be wary how they operate upon pellagrins.

Under the heading of treatment he writes no individual can correctly be said to be cured unless at least two years have elapsed during which the patient has shown constant improvement in health and no return of pellagrous symptoms. The author insists upon the importance of ascertaining whether any

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other disease is present, and advises a careful search for tubercle, malaria, entozoa and syphilis.

Everything should be done to increase the resisting power of the pellagrin. Arsenic in large doses is praised as the best drug, for instance, Fowler's solution, five drops two or three times daily, increased by one drop every day "until untoward effects begin. Some patients will continue up to twenty or twenty-five drops at a dose, but, as a rule, they do not stand well over fifteen drops three times a day." In addition to Fowler's solution by the mouth, cacodylate of soda (3-5 grains hypodermically two or three times a week) is also praised. Arsenic is to be continued from one to three months after all the symptoms have disappeared and is to be given again for about a month during the following spring.

Atoxyl, soamin and salvarsan are also commended and the author has used transfusion in three cases with temporary improvement, but two of the patients subsequently died and the third was not cured.

With regard to diet, the patient is to be fed as generously as his digestive tract will allow; meat, eggs, butter, and fats should be given in large quantities.

The final chapter of this interesting book considers the unknown cause of pellagra and the author states that he inclines towards Sambon's theory of infection.

[In spite of some errors of spelling of proper names and of Italian words, this would seem to be the best monograph on pellagra in the English language.]

F. M. Sandwith.

Etiology.

Bertarelli (E.). **Osservazioni Critiche a proposito del Problema Eziologico della Pellagra.** [Critical Observations on the Etiological Problem of Pellagra.]—*Gazz. d. Ospedali e d. Cliniche.* 1912. Aug. 4. Vol. 33. No. 93. pp. 961-963.

The author discusses the various theories and is of opinion that it is only the doctors on the spot, living in pellagrous districts, with eyes and brain specially concentrated on the disease, who can succeed in determining whether we have to deal with an intoxication or an infection. He pleads for such patient research as eventually revealed to us the mystery of malaria. He considers that the maize theory is supported by the recent work of Volpino and others, who showed that pellagrins exhibited a specific anaphylactic sensibility to the poison of diseased maize when it was injected subcutaneously.

F. M. S.

Nicholls (Lucius). **Tropical Pellagra.**—*Jl. Trop. Med. & Hyg.* 1912. Aug. 15. Vol. 15. No. 16. pp. 241-245.

The author reports nine cases occurring in the island of St. Lucia during a period of three months. He considers that the condition of the organs at two post-mortem examinations, "the extraordinary emaciation, the small haemorrhages or diapedesis

of red blood corpuscles throughout the organs, the lessened coagulability of the blood, the absence of much temperature or congestion of any organs, the presence of fatty degeneration—very similar in degree to what is seen in some cases of beriberi—and the condition of the tongue and gums, indicate that the disease is more nearly allied to such diseases as scurvy and beriberi than to any germ disease."

F. M. S.

Nicholls (Lucius). **Pellagra. "Sandfly Protozoon" *versus* "Zeist" Theory.**—*Jl. Trop. Med. & Hyg.* 1912. Oct. 15. Vol. 15. No. 20. pp. 305-306.

The author, writing from St. Lucia (West Indies), says that he is not an ardent supporter of either maize or insect-borne causation, but he objects to attempts "to force all factors to fit a preconceived theory."

He narrates the case of a middle-aged woman who by reason of an ulcerated leg was unable to work; having no friends to support her, she limped to a damp valley in the island and subsisted for some weeks upon "yams, plantains, farina and corn meal"; this mouldy corn-meal [? maize] was said by a negro not to be fit for pig food. She was found by the author to be suffering from typical pellagra—"dementia, dermatitis, sore mouth, diarrhoea, refusal of food, emaciation, shrinkage of liver and spleen and slightly lessened coagulability of the blood." During three years at least no other case of pellagra has been seen in this part of the island, but the author adds that the inhabitants rarely eat corn-meal.

F. M. S.

Sheppard (W. Sidney). **The Etiology of Pellagra.** [Correspondence.]—*Brit Med. Jl.* 1912. Dec. 28. p. 1773.

The author records the existence of pellagra in Singapore among the very poorest class who subsist almost entirely on rice. He states that maize can have nothing to do with its causation in the Straits Settlements and he doubts whether simulium occurs there.

F. M. S.

Lavinder (C. H.). **Certain Aspects of the Pellagra Question.**—*Southern Med. Jl.* 1912. Aug. Vol. 5. No. 7. pp. 476-482.

This is a paper read before the State Medical Association at Birmingham, Alabama, and deals mostly with the difficulties surrounding the etiology and the diagnosis of the disease.

F. M. S.

Devoto (Luigi). **Aetiologie und Klinik der Pellagra.**—*Wiener Med. Wochenschr.* 1913. Jan. Vol. 63. No. 1. pp. 20-28.

The author met with pellagra for the first time in 1899 when he was elected professor of medicine in Pavia. Finding that for

the last twenty years clinical physicians had neglected the study of the disease, and had resigned it to psychologists, hygienists and pathologists, he instituted a pellagra section in his clinic and claims that this is the first time this has been done since the Emperor Josef II created one in Legnano, near Milan, under the well known STRAMBIO. He maintains that most students of pellagra, unlike LOMBROSO, have neglected to study the clinical forms and the changeable course of the disease.

BIANCHI and AGAZZI's researches which show that in various types of pellagra, there is an increase of large mononuclear cells (twelve to eighteen per cent.) have been claimed by SAMBON as an additional proof of his protozoal theory, but the author points out that this increase is also seen in lymphatic leukaemia, chronic alcoholism and small pox, and he suggests that this is not evidence of protozoal infection, but rather of deficiency of nutrition. He insists upon the importance of discovering the earliest symptoms of the disease (heaviness of head, tendency to melancholia, apathy, restlessness, loss of flesh, debility, indigestion, flatulence, giddiness, etc.) during the winter preceding the first appearance of the eruption in March. It is precisely during the winter that the economic conditions of the peasant become worse and worse and his diet is gradually narrowed down to maize, often of inferior quality. The author is a confirmed upholder of the maize theory, though not necessarily of bad maize as LOMBROSO taught, and he quotes the work of BEZZOLA who since 1903 has been experimenting on guinea pigs and mice. He finds:

1. An exclusive diet of maize is an insufficient food for guinea pigs and they die after a few weeks.
2. If bran and vegetables or vegetables alone are added to the maize the diet is nourishing, but long continuance of this mixed diet may lead to a constant group of symptoms chiefly characterized by acute or chronic inflammation of stomach and intestines of the guinea pigs and by marked temporary loss of hair.
3. It is almost immaterial whether the maize is good or bad.
4. Mice on the other hand are not affected by a maize diet extending over twelve months.

LUKSCH repeated BEZZOLA'S experiments and found the same results with rabbits that the latter had seen with guinea pigs. The author is a strong disbeliever in SAMBON'S theory and considers that the arguments against it are much stronger than SAMBON'S criticisms against the maize theory. He also insists that the peasant shows signs of pellagra before the simulium appears in the spring months.

The author states that in eight Italian provinces there has been a diminution since 1889 of 75 per cent. of pellagrous individuals so that it is now difficult to find 100 typical cases during the pellagra seasons. He asks: "Is the simulium disappearing or is the protozoon ceasing to be infectious?" He concludes by maintaining that the diminution of pellagra in Lombardy is due to lessened consumption of maize, to edicts forbidding the use of bad maize and to improvement in the economic conditions, because a mixed diet has become habitual among the peasants and the fight against pellagra is now universal.

F. M. S.

SYMPTOMATOLOGY.

STIEFLER (Georg). **Das Fazialisphänomen in der Symptomatologie der Pellagra.** [The Facial Phenomenon as a Symptom of Pellagra.]—*Neurol. Centralbl.* 1912. Dec. 1. No. 23. pp. 1483-1488.

This is a study of a symptom described by NEUSSER and found to be present in most of the cases he studied in Austria and Roumania. It consists of mechanical excitability of the facial nerve and is sometimes called "Chvostek's symptom." It used to be thought that this symptom was peculiar to tetanus and according to FRANKL-HOCHWART, it consists of four degrees (1) spasmodic reflex of the whole face produced by tapping in front of the ear, (2) reflex of nostrils and corner of the mouth, caused by tapping under the zygomatic arch, (3) quivering of mouth only, (4) reflex induced by gentle stroking of the face.

In May and June of 1906 and 1907 the author examined 140 obviously pellagrous peasants of the Italian part of the south Tyrol, three-fifths men and two-fifths women, mostly aged between twenty and fifty. They were inmates of the Pellagrosarium in Rovereto and of the Pergine asylum, some chronic, others of recent development. Notes are given of fifteen cases in which the reflex was obtained, and eleven of them showed the quivering of the mouth muscles not always equally marked on the two sides. He saw no cases resembling tetanus which NEUSSER had done and he suggests that his different results may be due to this cause. He considers that the reflex cannot be of assistance in diagnosing doubtful cases of pellagra, because the phenomenon occurs in tetanus and in some other nervous diseases.

F. M. S.

BONDURANT (E. D.). **Terminal States in Pellagra resembling General Paresis, with Report of Five Cases.**—*Jl. Nerv. & Ment. Dis.* 1912. Nov. Vol. 39. No. 11. pp. 734-738.

Among fifty pellagrous lunatics studied in Alabama, the author has met with five who presented symptoms strongly suggestive of general paralysis. Syphilis had existed in one case; in the four others it was denied and in two of them the Wassermann reaction was negative. Three of the patients died but no autopsy seems to have been made. The author wishes to draw attention to the possibility of a typical general paralysis of the insane being of pellagrous origin.

F. M. S.

HOAG (D. E.). **Pellagra: Observations on some of its Nervous Manifestations.**—*Jl. Amer. Med. Assoc.* 1912. Oct. 19. Vol. 59. No. 16. pp. 1445-1446.

This paper, written by a neurologist entirely from the clinical point of view, was read in the section on Nervous and Mental Diseases at the last meeting of the American Medical Association. The author considers that the whole mental and neurological picture of the pellagrous lunatics whom he has seen is indicative

of a toxaemia, with a subsequent degenerative change in cells of the central nervous system, notably the cortex cerebri.

"The most definite changes in the spinal cord would point to an involvement of the lateral tract and cross pyramidal tracts. In general, the nervous symptoms most clearly resemble a neurasthenia interspersed with periods of excitement and delirium. Some cases, with mental defect, slurring speech, tremor of the facial muscles and of the tongue, suggest a general paresis."

F. M. S.

Wood (Edward J.). **"Pellagra *sine* Pellagra."**—*New York Med. Jl.* 1912. Aug. 3. Vol. 96. No. 5. pp. 218-220.

The author holds strongly the view that pellagra never occurs without some skin lesions and even urges that it would otherwise be impossible to diagnose this disease from sprue, dysentery and "many sub-tropical diseases which occur in the southern States." But he recognises that slight disturbances of the skin are often overlooked by both patient and doctor and he quotes the case of an old woman who had been under his constant care for two years; intractable diarrhoea, "typical pellagrous stomatitis," and symmetrical erythema around the anus, all preceded by some months a very definite furfuraceous desquamation of the backs of the hands, which occurred after the patient had been placed out of doors in the spring sunshine.

As Chairman of the Pellagra Commission in North Carolina, he draws attention to the fact that an early diagnosis is, as regards treatment, quite as important in pellagra as in tuberculosis.

F. M. S.

Snyder (J. Ross). **Pellagra in Children. (A Preliminary Report.)** —*Amer. Jl. of Diseases of Children.* 1912. Sept. Vol. 4. No. 3. pp. 172-179.

Letters addressed by the author to sixty physicians, apparently in the State of Alabama, where pellagra is well known, elicited replies which strongly suggest that children in towns enjoy immunity to a considerable degree. In answer to a question as to the youngest age when undoubted pellagra has been observed, three correspondents stated two months, four months, and five months, while others had never seen it earlier than at the age of thirteen or fourteen years. The answers of 31 correspondents show that pellagra is met with much more frequently after the eighteenth month.

Other answers tend to show that about ten per cent. of all pellagrins are children under fifteen years of age, and that, as a general rule, marked nervous symptoms in pellagrous children are rare, for the eruption may often be the only symptom prominently manifest.

One case is quoted of a pellagrous mother who nursed her child until the baby was six weeks old when she died. The infant was then fed on malted milk and developed pellagrous erythema several days after the mother's death. The author considers that the possibility of a pellagrous mother transmitting the disease by her milk has not been disproved.

F. M. S.

Pellagra in the United States.

Harrington (Arthur H.). **Pellagra in Rhode Island.**—*Boston Med. & Surg. Jl.* 1912. Nov. 21. Vol. 167. No. 21. pp. 731-733.

The scope of this paper, which was read before the Pellagra Conference at Columbia last October, is chiefly historical and shows that pellagra has existed undiagnosed at the State Hospital for the Insane at Howard since 1891. Until 1910, when the first case was recognised as pellagra, the lunatics were thought to suffer from "tropho-neurotic sunburn." Since 1910, there have been 37 cases diagnosed with 16 deaths.

The author relates five other cases which have occurred in the district, showing that pellagra is not confined in Rhode Island to the State Institution.

F. M. S.

Lavinder (C. H.). **The Prevalence and Geographic Distribution of Pellagra in the United States.**—*U.S. Public Health Rep.* 1912. Dec. 13. Vol. 27. No. 50. pp. 2076-2088. With 1 map, 3 charts and 3 tables.

A systematic attempt has been made by the Surgeon General of the Public Health Service to collect statistical information relative to pellagra in the United States. In answer to letters and cards sent out to individual practitioners and superintendents of hospitals, asylums and other institutions, replies have only been received from one-fourth. From eight of the Southern States, 4,657 answers have been received recording 15,870 cases during the five years, 1907-1911, with an average mortality of 39·1 per cent. This death rate, which is extremely high for pellagra, as judged by other countries, varies from 30·9 in South Carolina to 55·5 in Virginia. In the eight States the white patients more than doubled the coloured cases, but these figures do not include reports from the lunatic asylums. The charts show that there are more than double the number of cases in country districts as compared with towns, while 6,641 cases are reported among the poor, 5,359 are in moderate circumstances and 1,102 are believed to be "well to do." The comparatively large number of cases occurring in people of easy circumstances is a peculiarity of the United States, though the author quotes the saying of an Italian writer, Dalla Bona: "It does not always spare the palace." As in Italy the Americans find that there is constantly only one case in a house or family. The map shows very clearly that in all the States to the south pellagra is prevalent, that it occurs sporadically in those north of them and that it has not yet been recorded from nine in the further north and to the west.

The author considers that pellagra has already claimed during the last five or six years, not less than 30,000 victims in some thirty States and that the matter has reached the dignity of a public health question of national importance.

F. M. S.

Pellagra in Illinois. Condensed Report of the Illinois Pellagra Commission.—*Arch. Internal Med.* 1912. Aug. 15 & Sept. 15. Vol. 10. Nos. 2 & 3. pp. 123-168 & 219-249.

Ormsby (Oliver S.). [Medical Secretary of the Commission.] **Pellagra.**—*Jl. Cutaneous Dis.* 1912. Oct. Vol. 30. No. 10. pp. 589-607.

Pellagra was first recognised in the State of Illinois in June 1909, in an asylum, and with commendable promptness the Governor of the State, before the end of the year, appointed a commission of ten doctors to investigate the disease. Excluding cases remembered before 1909 and all doubtful cases reported since that year, they now believe that there have been at least 500 pellagrous patients in this one State, with a mortality in ten institutions of 46·3 per cent. More than half the cases occurred at the Peoria State Hospital which had a total population of 2,100, and 8·4 per cent. of the lunatics showed definite symptoms of pellagra.

Yet "none of the employees suffered from the disease in spite of the fact that they were exposed fully as much to the bites of insects and drew their food and water supply from exactly the same source as the patients. This freedom from pellagra on the part of physicians, nurses, attendants and other employees, has been absolute in all the institutions."

The skin lesions of more than 200 patients were carefully examined and coincided as regards site and symmetry with the accounts of previous writers, but less atrophy was noted than in European and Egyptian writings. In a large number of sections made—

"the general picture was that of an angio-neurotic process, and resembled to a marked extent that seen in multiform erythema. The most marked change was noticed in the superficial part of the corium, almost all infiltration occurring in the pars papillaris. The specific findings are as follows: With a low power, the stratum corneum was thickened, the stratum granulosum and rete practically normal. The upper portion of the corium showed inflammatory reaction and the connective tissue appeared oedematous. With a high power the hyperkeratosis was seen to be well marked. Here and there, areas of parakeratosis were present, as evidenced by the presence of nuclei extending to the upper layer of the stratum corneum. Many pigment granules were present. The rete was practically normal, except in places where its integrity was interfered with by infiltrating cells. In the papillary layer cellular infiltration was quite marked, particularly in the region of blood vessels. Collagen and elastin were present, the former showing oedematous changes. The deeper parts of the corium were comparatively normal. In parts of the papillary layer elastin was absent."

The Commission do not feel justified in making any definite statement regarding the nervous and mental symptoms of pellagra and they doubt the existence of any "pellagrous insanity." But they emphasize the great and unexplained susceptibility of the chronic insane to pellagra.

Detailed examinations of faeces, blood, and urine proved negative and the report states that no support could be found for regarding pellagra as a complication of amoebic dysentery and "nothing whatsoever suggestive of a protozoal infection was encountered." Seven post-mortem examinations made showed

fatty degeneration of liver cells, non-amoebic intestinal ulceration in three instances, more or less interstitial nephritis, and pigmentary changes in the heart muscle. The complete examination of the spinal cords is not yet published but the summary states that—

"the nervous system presents a picture of axonal chromatolysis involving especially the Betz and larger pyramidal cells of the precentral convolutions and the cells of the nuclei in the cerebellum, pons, medulla and cord, as well as the posterior root and sympathetic ganglia."

Attempts to transmit pellagra to monkeys and guinea pigs completely failed.

Complement fixation tests by Noguchi's method gave "results which cannot be regarded as specific at present. Negatives with positive cases and positives with normal sera have been encountered too frequently to permit of any interpretation."

Anaphylactic tests by Von Pirquet's method, substituting maize extracts for tuberculin, proved negative. An interesting experiment devised by Captains Siler and Nichols tends to discredit the causal relationship of maize. Two cottages in the Peoria asylum, each fitted for about sixty patients, were filled with non-pellagrous chronic lunatics. One cottage was placed on a generous maize diet, the other on a maize-free diet. At the end of twelve months the maize eaters had four certain and one doubtful case of pellagra, while the maize-free group included five certain and five doubtful cases of pellagra. As an argument in favour of the deficiency of nutrition theory it was found that an increase of meat to the dietary diminished the number of pellagrous cases, while they were increased in another asylum when the meat supply was reduced. There are said to be 65 species of simulium in the world; 15 have been found in the United States, nine of which are known to occur in Illinois.

"The relation of simulia to pellagra, hypothecated by Sambon, finds but little support from the researches we have been able to make. The particular variety, *S. reptans*, which he claims to be of world-wide distribution, is said by Professor Forbes (State Entomologist) to be unknown in North America as yet except in Greenland."

Conclusions.—

"1. Pellagra is a disease due to infection with some living micro-organism.

"2. A possible habitat for this parasite in man is the intestinal canal.

"3. Deficient animal protein in the diet may constitute a predisposing factor in the contraction of the disease.

"4. The number of cases of known pellagra renders this disease a decided menace to the public health of this State.

"5. Careful search for and investigation of suspected cases outside the State hospitals for the insane is extremely desirable in view of experience elsewhere."

[The complete report of this Commission is shortly to be issued. It will apparently be the most valuable and original contribution to the literature of pellagra which has yet appeared in the United States.]

F. M. S.

Babcock (J. W.). **How Long has Pellagra Existed in South Carolina? A Study of Local Medical History.**—*Amer. Jl. of Insanity.* 1912. July. Vol. 69. No. 1. pp. 185-200.

In December 1907 pellagra was officially recognised as being present in the State Hospital for the Insane, Columbia, South Carolina, and in 1910 this diagnosis was made in 135 cases or 20 per cent. of the admissions. The author has made a diligent search through all the case papers of former years and though the records are often imperfect he believes that ever since the hospital was opened in 1828, there has been an elusive and puzzling disease which may well have been pellagra, sometimes complicated by scurvy. In the early days of the hospital the prevailing fatal diseases were recorded as chronic diarrhoea or dysentery, while of later years he has discovered vague diagnoses such as inanition, exhaustion, consumption and marasmus. He suggests that under these terms some cases of pellagra may have been concealed.

F. M. S.

Lee (Roger L.). **A Case of Pellagra that had not been outside of New England for Eighteen Years.**—*Boston Med. & Surg. Jl.* 1912. Sep. 26. Vol. 167. No. 13. pp. 435-436.

The patient, a white dry-goods clerk, after some indigestion in the spring months for the past three years, began to suffer in May, 1912, from red, burning hands, for which no cause could be assigned; this was followed by loss of appetite, weight and strength, epigastric discomfort, salivation and diarrhoea. On examination at the Massachusetts General Hospital in July, he was found to have symmetrical erythema of face, hands, wrists and nape of neck. Normal white skin was seen under a ring on a finger. His tongue and anus were abnormally red and his mental state was confused and depressed, with occasional excitement. His loss of weight amounted to 59 pounds in a year. He rapidly improved on good food. His wife (who was in good health) and he lived on ordinary diet under fairly hygienic circumstances. "They ate ordinary commercial wheat flour and denied absolutely eating maize or other maize products." It could not be discovered that the patient had been exposed to any simulium bite. "The only possible peculiarity of diet for some years past was that two years ago the patient and his wife bought a large amount of potatoes, and ate a great deal of that particular supply during one winter, but not to any great excess or to the exclusion of the ordinary mixed diet."

[Sporadic cases have already been reported from Massachusetts and other northern States.]

F. M. S.

Pellagra in Italy and other Countries.

Lavinder (C. H.). **Pellagra in Italy. A Note on the Prevalence during the Years 1881-1899-1910.**—*U.S. Public Health Rep.* 1912. Nov. 1. Vol. 27. No. 44. pp. 1778-1779.

This is a copy of the table of official statistics in Italy during the three years named, taken from the *Rivista Pellagrologica*

Italiana of July 1912. The Rivista is a publication which appears twice a month and is the official organ of the Permanent Committee of the International League against Pellagra and of the Pellagra Commissions of Italy. The total number of pellagrous cases in 1881, when the disease in Italy reached its zenith, was 104,067; in 1899 there were 72,603 cases; and in 1910 there were only 33,869 cases in spite of a steady increase of the rural population. Whether the figures are accurate or not there is ample reason for believing that the disease in Italy is certainly decreasing both in number of cases and virulence. The decrease evidently began before July, 1902, when the national law against pellagra became effective.

F. M. S.

Goodwin (E. S.). **Pellagra in Hawaii: Report of a Case.**—*New Orleans Med. & Surg. Jl.* 1912. Aug. Vol. 65. No. 2. pp. 120-137.

The author states that the American Indian, who has grown and eaten maize for generations, is free from pellagra. He reports the case of a female native who has never been out of Hawaii and who has never eaten maize in any form. He believes that this is the first case which has been reported from the islands of the Pacific. The patient's illness began in November 1910, some fifteen months before this report was written. The prominent symptoms at the onset were: burning pain in the epigastrium, pyrosis, constipation with occasional diarrhoea. In the summer of 1911 to these were added loss of memory and appetite, "some fever," great thirst, ringing in ears, epileptiform attacks, which have continued. In the autumn "dermatitis covered back of hands, left elbow, feet, legs, cheeks and forehead. There was oedema and subsequent exfoliation. Then pigmentation of affected areas, more pronounced upon hands, feet and legs than elsewhere." In December 1911 she became bed-ridden with loss of knee jerk and tenderness on pressure over spinal region, also "extensor muscles of arms, hands, feet and legs much atrophied with spastic paraplegia."

The author considers "the case is one of unmistakable pellagra."

[The photographs of the patient are not published with the paper.]

F. M. S.

Diagnosis.

Rauenbusch (Hugo). **Zur Frage einer spezifisch-diagnostischen Reaktion bei Pellagra.** [A Specific Diagnostic Reaction in Pellagra.]—*Deut. Med. Wochenschr.* 1912. Nov. 14. Vol. 38. No. 46. pp. 2169-2171.

Writing from Czernowitz, the author draws attention to former papers which seem to prove the deleterious effects on white mice and guinea pigs of an exclusive maize diet, whether of good or bad quality, so long as the animals are exposed to sunlight. He suggests that an exclusive diet of other cereals, such as rice,

millet or wheat, might under similar circumstances produce the same phenomena. He states that some years ago he cured mice which were suffering from the effects of maize and sunlight by keeping them in darkness, though the diet was unaltered. Excluding light from the skin of pellagrous lunatics by means of darkened rooms, red windows, ointment and bandages, seemed to have favourable results upon the few individuals on whom these experiments were tried.

In consequence of the view held by many Italian writers that pellagra is an infectious disease with some micro-organism in the blood, and because of Tizzoni's claim that he has found the "streptobacilli" of pellagra in the blood, cerebro-spinal fluid, and organs after death, the author thought it right to re-investigate this point. He now states that he was unable "in several hundred examinations to isolate any germs in any single instance." He complains that when he invited Tizzoni to send him cultures of his bacillus, he received nothing but good advice. In about 100 cases he was able to satisfy himself that there is no diagnostic value in Tizzoni's belief that pellagrous blood serum will agglutinate minute quantities of rabbit's blood without haemolysis. He is equally sceptical about the serum reactions described by Gosio and Bass. Some experiments of his own to try and produce a cutaneous or ophthalmic or subcutaneous reaction on pellagra patients by means of different aqueous extracts of maize also proved useless.

His conclusions are:—

In the blood and in the organs of pellagrous patients no specific germs can be found by our present bacteriological methods. The blood of such patients, examined by our present cultural methods, proves to be exceptionally free from germs.

As tested by our present serological methods, no anti-bodies nor material exist in the serum of pellagrous patients which are peculiar to pellagra.

The etiology of pellagra is photodynamic and the disease is produced by a diet of good or bad maize, which however can only display its deleterious action when the patient is brought under the influence of sunlight. It is possible that other forms of diet (rice, millet, &c.) might produce similar disease under the influence of sunlight.

F. M. S.

Tizzoni (Guido). **Ueber die immunitäre Reaktion des Blutes bei der Pellagra. Vorläufige Mitteilung.** [The Immunity Reaction of Blood in Pellagra.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1912. Dec. 4. Vol. 67. No. 3. pp. 175-177.

The author continues an account of his researches in Bologna and claims to have proved that (1) ordinary laboratory bouillon exercises no influence on the red blood corpuscles of the rabbit, which form a sediment and remain intact a comparatively long time without changing; (2) the blood serum of healthy man added to similar bouillon and rabbit's blood, agglutinates the blood cells to a slight degree and always haemolyses them (normal hetero-agglutination and heterolysis); (3) the blood serum of pellagrous patients on the other hand, when similarly treated, agglutinates the blood cells much more quickly and more

thoroughly but never develops a haemolysing action. He maintains that in the blood of pellagrous patients there is present an anti-haemolysin which counteracts the haemolytic capacity of normal heterolysin.

[See RAUBITSCHEK's paper above.]

F. M. S.

RONDONI (Pietro). **Sulla Ipersensibilità dei Pellagrosi al Mais.** [The Hypersensitiveness to Maize of Pellagrous Individuals.] —*Lo Sperimentale.* 1912. Sept. 26. Vol. 66. No. 5. pp. 447-472.

The author set himself the task of controlling on human beings the recent work of VOLPINO, MARIANI, BORDONI and ALPAGO-NOVELLO, and of noting the general and local phenomena which occurred after the injection of maize intramuscularly. He obtained his "subjects of experiment" from pellagrous convalescents from Città di Castello, from lunatics of that town and from a hospital in Florence.

Whereas VOLPINO experimented with maize bought from a merchant for feeding animals he thought it wiser to procure maize directly from the domestic store of pellagrous victims. After a careful disinfection of the skin the injections were made in the thigh or buttock, usually preceded by a VON PIRQUET test to see if the subject reacted to tuberculin. Notes of all the experiments are given.

In all 33 pellagrous and 30 non-pellagrous individuals were injected with an extract of maize, while eight of the pellagrous also had an injection of an infusion made from wheat or chestnuts.

The author never found a violent reaction as described by other writers and his conclusions are: (1) In comparison with the non-pellagrous, recent cases of pellagra and convalescents from that disease exhibit more often and more certainly a definite reaction (slight rise of temperature, malaise, headache, excitability, sleeplessness) following on the subcutaneous injection of an extract of spoiled maize.

(2) This enhanced sensibility of the pellagrous *may be* interpreted as an anaphylactic reaction to some unknown and therefore undefined factors of the maize extract and may be in connection with maize diet by some kind of sensibility of the organism through the alimentary tract.

F. M. S.

HIRSCHFELDER (Arthur D.). **Gibt es besondere fluoreszierende Substanzen im Serum bei Pellagra?** [Fluorescent Substances in Pellagrous Serum.]—*Centralbl. f. Bakt.* 1. Abt., Orig. 1912. Oct. 29. Vol. 66. No. 7. p. 537.

This paper from the Psychiatric Clinic of the Peoria General Hospital, Illinois, and from the Medical Clinic of Johns Hopkins University, is in antagonism with RAUBITSCHEK's* idea that a

* RAUBITSCHEK. Zur Kenntnis der Pathogenese der Pellagra. *Centralbl. f. Bakt.* 1. Abt., Orig. 1911. Vol. 57. p. 193.

fluorescent substance is necessary for a skin disease, sometimes fatal, which occurs in white mice which have been fed on maize and also exposed to sunlight.

The author states that in 1910, a year before RAUBITSCHEK'S paper appeared, he had searched for such fluorescence in the blood serum of five patients suffering from acute and pronounced pellagra. He found that the serum of normal individuals showed a certain fluorescence and that the serum of pellagra patients exhibited only a similar fluorescence.

F. M. S.

TREATMENT.

NICOLAIDI (Jean). **New Treatment of Pellagra, with the "Organo-Mineralized Radio-Activated Serum" of Dr. Jean Nicolaidi, Paris, France.**—*Southern Med. Jl.* 1912. Aug. Vol. 5. No. 7. pp. 464-475. With 14 photographs.

After working at pellagra with his father, a physician in Roumania, the author set himself the task of (1) neutralizing the "toxine" or the "virus" which pellagra produces in the organism, (2) awakening the intracellular activities and the reparative forces tending to remedy the deficiencies caused by the disease. For this purpose he adopted an "artificial organo-mineralized serum" which he rendered radio-active by a special process. He states that he "was able to unite all the elements extracted from the horse serum by evaporation in the vacuum, which he treated subsequently by means of a solution saturated with carbonic acid containing all the organic and mineral salts of the blood plasma." In 1910 he was appointed by the Roumanian Government to conduct some experiments in a hospital at Craiova and these were favourably reported on by the chief physician of the hospital and the senior physician of the lunatic asylum who wrote:—"After a certain number of injections (20 to 25) some of the patients were cured, some considerably improved, [results] which have not been obtained by all the different treatments applied during months in the asylum."

In the autumn of 1911 the author was appointed by the Italian Government to undertake similar experiments in Udine, under the control of Professor ANTONINI, the well-known director of the lunatic asylum there.

The number of cases treated is not mentioned, but the Italian physicians (GRILLO and MAJ) reported that nearly all the patients treated "have shown a marked improvement in a short length of time." Most of the patients were able to resume their occupations which they had long since abandoned. Professor ANTONINI considered that "we have found the means of fighting against pellagra at the moment when energetic methods may be necessary." Professor BLANCHARD saw some of the patients in Craiova when the experiments were finished and stated in a lecture at the Academy of Medicine in Paris that the results obtained were most remarkable.

F. M. S.

Reports.

Lavinder (C. H.). **The Association for the Study of Pellagra. A Report of the Second Triennial Meeting held at Columbia, S.C.,** [South Carolina], **Oct. 3-4, 1912.**—*U.S. Public Health Rep.* 1912. Nov. 1. Vol. 27. No. 44. pp. 1776-1778.

At this congress 65 communications were presented on the various phases of pellagra and covered the etiology, epidemiology, statistics, American history, diagnosis, laboratory investigations, clinical features, treatment and miscellaneous aspects of the disease. One of the resolutions stated that the ultimate cause of pellagra is unknown but that in view of the incrimination of spoiled maize "measures should be taken by the proper authorities to prevent its sale and consumption as food."

Hope was expressed that the Congress of the United States may appropriate sufficient funds for the continuance and extension of anti-pellagra research.

Some of the delegates entertained "a feeling, almost a conviction, that the disease is of an infectious nature and probably insect-borne."

Dr. C. H. Lavinder of the Public Health Service was elected the new President of the Association. [Pellagra students are anxiously waiting for the published Proceedings of this meeting, though none of the contributions seem to have been epoch-making.]

F. M. S.

PLAGUE.

Plague in the Far East.

Lancelin. **La Peste de Mandchourie. Sa Prophylaxie.**—*Arch. de Méd. et Pharm. Nav.* 1912. Nov. Vol. 98. No. 11. pp. 353-374.

It seems certain that the outbreak began in Eastern Mongolia in September 1910, and was caused by an epizootic in the tarabagans. The trappers brought the infection to the frontier railway station Manchuria, where the first death occurred on the 25th of October, 1910, followed by 37 more in the space of six days. Plague rapidly spread along the railway line since the Chinese had not informed the Russian authorities of the occurrence. Hence it was not long before it reached Tsitsikar. Kharbin was infected on November 9th. On December 31st two cases were discovered in the train going from the junction between the Russian and Japanese lines at Kouang-Tcheng-Tse, which had become a plague centre shortly before, to Mukden. The first death at Mukden took place on January 3rd, 1911. From this date the scourge spread with great rapidity. The whole district between Kharbin and Mukden was ravaged. Deaths occurred by hundreds a day at Kharbin and the neighbouring Chinese town Fouchiatien, with populations of 30,000 and 35,000 respectively.

Sketch Map of Manchuria and Neighbourhood.

[From the map illustrating Lancelin's paper. The French spelling for names of towns and rivers, &c., is retained.]

Notwithstanding the stringent precautions taken by the Japanese railway authorities on their South Manchurian line, the epidemic extended to the Kouang-Toung peninsula, and reached Dalny. The Korean railway line also carried the infection.

The pestilence travelled south, and defying the Sanitary Cordon at the Great Wall, and the quarantine station Chan-Hai-Kouan, it attacked Tientsin, Pekin, and Pao-Ting-Fou in succession. Going eastwards it visited Tsinan-Fou, and crossing the Yellow River invaded Tchefou. South of the Great Wall the epidemic raged with less violence than in Manchuria, and it began to subside in February. In Manchuria on the other hand, its ravages continued till the middle of April, when they were brought to a sudden close, but not before the depopulation of whole towns.

The administrative organization for the control of the epidemic was subdivided.

Russia organised the preventive measures in Kharbin, and guarded the trans-Siberian railways and the frontiers along the Amour and Oussouri rivers. The Chinese Government protected Petchili by a sanitary cordon along the Great Wall and by surveillance of the Mukden-Pekin railway. The Japanese were responsible for the sanitary administration of the South Manchurian Railway, Korea, and the peninsula of Kouang-Toung. In the foreign concessions the plague campaign was carried out by the country concerned.

At Kharbin the administration, the head of which was Dr. Dautchakoff of the Russian Navy, was granted full powers. The city was divided into four sections, to each of which a personnel of medical officers, inspectors, attendants and police was allotted. The inhabitants of each section were obliged to wear a distinguishing brassard, and no communication was allowed between them. A military guard was placed on the bridge joining Kharbin with Fouchiatien. Every house was visited daily; the residents were inspected and their temperatures were taken. Suspicious cases were immediately reported to the head office; the patient was caused to expectorate on a piece of paper, the outside of which bore his name and address and date, which was sent to the laboratory. In febrile cases, when neither cough, expectoration, nor physical signs were present, bacteriological examination of the blood often allowed a diagnosis of plague to be made. Since admission to a plague hospital was equivalent to a sentence of death, the greatest care was taken to prevent any errors.

There were many railway wagons available at Kharbin unused since the Russo-Japanese war, which were of great service for the isolation of suspected cases and of contacts. A thousand of these were soon required. Each wagon could accommodate 20 people. Suspicious febrile cases with a temperature of 37·9° C. and over, without cough or sputum, were placed two in a wagon; if the temperature was 37·2° C. four were allotted. Apparently healthy contacts were accommodated by twenties to each wagon. The employment of the thermometer was of the greatest value, for by it 80 per cent. of the plague attacks were discovered.

All the public buildings were converted into plague hospitals.

Treatment with anti-pest serum or salvarsan was without success.

The patients were supplied with masks, and everyone who was employed on plague duties was inoculated with HAFFKINE'S vaccine, and wore mask, goggles, overalls, wash-leather gloves, and wading boots. The mask contained a pad of wool moistened with formalin. Notwithstanding these precautions, many of the ambulance transport staff fell victims to the disease.

There were numerous cold storage railway vans at Kharbin which proved invaluable for the sterilisation of clothes and bedding by simply conducting steam from a locomotive boiler into their interior.

Plague corpses were incinerated. Some even were exhumed for the purposes of cremation. It is noted that the coolies who were employed on this work escaped infection, although "*cultures magnifiques*" of *B. pestis* were obtained from the earth clinging to the shrouds.

A military cordon was placed around the city, from which no egress was permitted. On the railway the conveyance of 3rd and 4th class passengers was stopped, and a searching medical inspection of the others was made. When a case of plague was discovered, it was removed to hospital, and the passengers who were travelling in the same carriage were kept in quarantine in it under a military guard for five days. A quarantine station was established at Manchuria for passengers bound for Europe and one at Pogranitchnaia for those going to Vladivostock.

The Chinese broke with their ancient traditions in their campaign against the plague which they carried on outside the foreign spheres of influence. The examination and cremation of corpses were permitted for the first time in their history. They followed the Russians in their defensive measures. In deference to public opinion they allowed one hospital at Fouchiatien to be conducted on native methods. It was closed at the end of a week, but in that time 18 of the 19 native doctors lost their lives.

At Mukden the Chinese acted with energy. They destroyed with fire about 300 plague houses.

Notwithstanding a Chinese guard of 750 soldiers posted along the Great Wall, a quarantine station at Chan-Hai-Kouan, vaccination and 5-7 days quarantine of all passengers from infected areas, the pestilence spread to all the large cities of Petchili. Tientsin was attacked on January 19. Precautionary measures similar to those carried out at Kharbin, were vigorously pursued. All the European troops were inoculated and confined to barracks. These provisions were effective, for only 61 deaths occurred, all in the Chinese city.

The Japanese were very thorough and autocratic in their endeavours to arrest the progress of the epidemic in their possessions. The results were remarkable, for though Dalny was infected on January 11, yet 22 days later the plague was stayed. There were 38 cases only in that town, and altogether 207 in the peninsula of Kouang-Toung.

A cordon of troops along the frontier, and 5 days quarantine of all passengers, together with a bacteriological examination of

their blood, at the frontier railway station, Niou-Wiju, afforded almost complete protection to the peninsula of Korea.

C. Birt.

Murata (N.). **Die epidemiologischen Beobachtungen anlässlich der Pestseuche in der Südmandschurei, und zwar im Kaiserlich japanischen Verwaltungsdistrikte.** [The South Manchurian Plague Outbreak in the Districts under Japanese Administration.]—*Zeitschrift f. Hyg. u. Infektionskrankheiten.* 1912. Dec. 20. Vol. 73. No. 2. pp. 245-252.

The outbreak of pneumonic plague in north and south Manchuria in the winter 1910-1911 spread over an area of about 240,000 square miles, and caused the loss of more than 40,000 lives. The Japanese spent a million and a half yen in preventive measures, consequently only 228 deaths occurred in the parts under their jurisdiction, which comprise the province of Kuantung and the districts through which the South Manchurian railway passes.

The tarabagan, *Arctomys bobac*, which inhabits the Baikal Mountains on the north of Manchuria, is liable to plague epizootics according to the investigations of Zabolotony: its fur is in great request. On the approach of winter the men employed in hunting these animals were proceeding south from the most northerly situated station on the S. Manchurian railway, Changchun, at the rate of nearly a thousand a day. Besides these there was a number nearly as large of travellers on foot. Pneumonic plague broke out among these men, who scattered the infection broadcast on their journey to their homes.

In the districts controlled by the Japanese the first cases of plague were discovered in a train coming from Changchun on December 31st, 1910. All the other passengers, 149 in number were at once sent back to Changchun and there isolated. The Japanese were not unprepared. They had organised all the resources at their disposal, which included civil, military, naval, and railway staff and equipment. 69 medical officers, 29 assistants, 414 police officers, and about 2,000 others were employed in the campaign against the pest.

The preventive measures comprised, inspection of all travellers by railway or other means, and their detention in isolation barracks built for the purpose, for 7 to 11 days; quarantine of, and rat destruction in, all boats and shipping entering the ports of Kuantung; house to house visitation; rat destruction,—though 169,025 rats were examined between November 1910 and December 1911 none were infected; burning of plague houses.

28 plates were exposed at distances up to 110 cm. from the faces of plague patients while they were in the act of coughing; colonies of *B. pestis* appeared on seven. Guinea pigs could not be infected in this manner. Plague bacilli in thin layers on glass were killed by direct sunlight in one hour, and in six hours if the sky was overcast. When they were placed on thick hempen sacking they resisted 14 hours sunlight and 20 hours diffused day light.

One hundred-thousandth of a loop of a culture of the Manchurian plague bacillus causes the death of the tarabagan when inoculated beneath the skin. One millionth of a loop kills rats and guinea pigs, and one thousand millionth causes a fatal infection of mice.

C. B.

Uthemann. **Wie begegnete des Schutzgebiet Kiautschou der andringenden Pestgefahr?** [Plague Prevention at Kiautschou.]—*Arch. f. Schiffs- u. Trop. Hyg.* 1912. Dec. Vol. 16. No. 23. pp. 790-807. With map.

The German Protectorate of Kiautschou is situated in the Province of Shantung, in which the ravages of plague caused 3,000 deaths during the epidemic of 1910-11. One third of these occurred in the port Tschifu, which has a large trade with Kiautschou. The German Protectorate, however, escaped the scourge.

In January 1911, as the epidemic spread through Shantung, watchful supervision was kept over the places of ingress into the German Protectorate. Finally all avenues were completely closed for more than a month. Inoculation was employed in about 200 instances only.

C. B.

Deutmann (A. A. F. M.). **De Pest in Karangloo in de maanden Mei, Juni en Juli 1911.** [Plague in Karangloo, Java, in May-July, 1911.]—*Geneesk. Tijdschr. v. Neder.-Indië.* 1912. Vol. 52. No. 4. pp. 431-511.

In this *Bulletin*, No. 2, p. 62, is a note on the outbreak of plague which occurred in Java in the year 1911. Deutmann gives details of that part of the epidemic which affected the district of Karangloo, of which he had sanitary control. This district covers an area of 396 square kilometers and contains a population of about 100,000 persons. 278 cases of plague were notified, with a death rate of 88 per cent. The origin of the outbreak was traced to the port of Soerabaya, whither plague had been conveyed through the agency of rats in January 1911. Sporadic cases occurred inland shortly afterwards, but the disease did not assume epidemic proportions until the months of May to July. 66 per cent. of the 278 attacks investigated by the author were of the bubonic type; 32 per cent. were septicaemic; and 2 per cent. were pneumonic. 62 per cent. of those affected with bubonic plague suffered from inguinal gland enlargment of both sides, except in six instances: 27 per cent. had swelling of the axillary glands; and in 14·5 per cent. the bubo was situated in the neck. 15,683 persons were inoculated with either Haffkine's prophylactic, or with a similar vaccine prepared by Nijland in the laboratory at Weltevreden. Plague attacked 64 inoculated, and 214 uninoculated people, the mortality amongst the former being 82·8 per cent., amongst the latter 89·7 per cent. Roux and Yersin's anti-pest serum was used therapeutically in 14 cases, of which eleven died; 20 cubic-

centimetres were injected subcutaneously twice or three times. The prophylactic administration was successful in the case of a mother who nursed her son stricken with pneumonic plague.

In 82 houses where plague was reported, 98 dead and 42 living rats were discovered and in 128 houses adjoining these, 66 dead rats were collected. Only three dead rats were found on searching 19 houses situated in plague-free localities.

C. B.

DE VOGEL (W. T.). **The Connection between Man and Rat in the Plague Epidemic in Malang, Java, in 1911.**—*Far Eastern Assoc. Trop. Med.: Trans. Second Biennial Congress, held at Hongkong,* 1912. pp. 147-149. With 10 plates.

At the time when the number of plague cases was at its highest, no house rats could be produced by the inhabitants. By methodical search in the houses where plague had occurred, however, rat shelters were discovered in the bamboo poles which enter into the construction of Javanese dwellings. Both living and dead rats were found in most of the plague houses. Sometimes the nests were empty, sometimes they contained the mummified remains of these rodents. A plate is given showing dead rats in their nest made in a bamboo ceiling immediately above the bed of a man who succumbed to plague. Living rats harboured the *Xenopsylla cheopis*. In Malang, the number of rats was less in plague houses than in those free from infection. The inference is clear that the number of rat-fleas without their natural host was greater in the infected dwellings. If all the rats had died of plague the fleas would forsake their dead bodies and seek refuge on man, to whom they would convey the disease. For the construction of rat-proof houses the bamboos are split, or if whole bamboos are used, the ends are sealed with cement, or are capped with metal.

C. B.

PLAGUE IN HAVANA.

VON EZDORF (R. H.). **The Occurrence of Plague in Habana and the Measures adopted for its Control and Eradication.**—*U.S. Public Health Rep.* 1912. Oct. 18. Vol. 27. No. 42. pp. 1697-1702.

Three cases of plague, resulting in two deaths, occurred in Havana, Cuba, between July and September 1912. There was increased mortality among rats at the time of the outbreak, but of 291 examined, none were found to be infected with plague. Up to the date of the report the inspection of 8,166 rats had not led to the discovery of the disease among them.

The suppression of the outbreak is attributed to the early recognition of the infection. The buildings in Havana are rat-proof to a great extent. The campaign included rendering rat-proof all houses, stores, etc., which had not been protected, the collection of garbage in rat-proof receptacles, the rapid removal of rubbish, the destruction of rats by traps and poison, and the disinfection of infected houses.

103 vessels were fumigated by burning 3 lbs. of sulphur per 1,000 cubic feet, or 4 lbs. per 1,000 cubic feet of cargo on board. The amount of sulphur to be used was rapidly calculated by allowing three pounds for each ten tons of gross tonnage. Not more than 10 lbs. of sulphur was burnt in one pot, and the fumigation was continued for 12 hours. Hawsers were freshly tarred for 4 feet, and rat-guards were applied.

Seven days quarantine was required of passengers who had been residing in the infected area.

C. B.

Guiteras (J.). **Three Cases of Bubonic Plague in Havana.**—*Jl. Amer. Med. Assoc.* 1912. Nov. 16. Vol. 59. No. 20. pp. 1780-1784.

The author, who is Director of Health of Havana, gives a full account of the cases of plague recorded in the foregoing paper. An epizootic occurred among rats limited to three blocks of buildings close to the wharves, which were chiefly used as grain and food stores, at the beginning of June, 1912. At the end of the month this appears to have subsided, as few dead rats were found. On July 2nd a man who resided in this area died of plague, though it was not notified under that heading. Three other cases were reported from the same quarter of the city, two of which ended fatally. One of the patients who succumbed was treated on the 9th, 10th, and 13th days of the disease with anti-pest serum. A pustular eruption broke out on the 10th day.

The people responded with alacrity to calls made to them to remove refuse, and to render their residences and stores rat-proof. For many days the amount of sweepings and rubbish carried out to sea in lighters increased from the normal 450 tons to 2,000 tons.

Up to September 3rd, 17,974 rats had been destroyed; a premium of five cents per head being offered. 8,909 of these were examined, but none were infected.

The author has been breeding *Loemopsylla cheopis*, the common rat-flea of Havana, in his laboratory for several years. Their numbers diminish in summer. They are least numerous on rats in June.

C. B.

Pneumonic Plague.

Raynaud. **Six Cas de Peste Pneumonique dans la Banlieue d'Alger.**—*Rev. d'Hyg. et de Police Sanitaire.* 1912. Aug. 20. No. 8. pp. 861-867.

In the year 1911 there was an epidemic of bubonic plague at Doukkala in the interior of Morocco, resulting in the loss of 10,000 lives. Cargoes of hides from the inland districts are embarked at the Moroccan ports and reach Algiers by sea. Ruisseau is a suburb of that city in which are located tanneries, abattoirs, and stores which are overrun by rats, among which there had been considerable mortality. In July, 1912, a boy who had been employed daily in rat-catching, succumbed to plague

in its pneumonic form. His father, mother and a nurse were infected by him and died. The mother conveyed the disease to her sister, and the nurse to a friend. The onset in all was sudden and was marked by rigors, vomiting, and cough with blood-stained sputum; the signs of pneumonia were obscure until shortly before death which took place in 2 or 3 days. One of the patients who was treated by the intravenous and hypodermic injection of anti-pest serum lived some hours longer than the rest. Prophylactic administration of the same remedy did not avert the attack in two cases. In the campaign against the rats which followed, not a single one was found to be infected out of 1,610 examined. The poison "Grosboisine" was used for their extermination. Guinea pigs were placed in the house where the plague appeared, but they remained healthy.

The author refers to a family epidemic of pneumonic plague which occurred in 1903. A girl contracted the malady while working at a mill in Algiers, and carried it to her home where her father and mother also fell victims to it.

C. B.

Rau. **Eine Lungenpestepidemie in Südbrasilien (in Santa Maria, Rio Grande do Sul.)**—*Deut. Med. Wochenschr.* 1912. Dec. 5. Vol. 38. No. 49. pp. 2314-2315.

Sporadic cases of bubonic plague have occurred at Santa Maria for four years. The infection was brought from India to the ports Rio Grande and Alegre, thence it spread up country, to Santa Maria, a town of about 18,000 inhabitants. Plague never assumed epidemic proportions in that town, and was preceded by mortality among rats. Suddenly the type changed from the bubonic to the pneumonic form. At the end of July, 1912, one of the occupants of an insanitary bake-house, where 200 dead rats were found, was attacked with pneumonic plague and died. 17 persons, either directly or indirectly, contracted the disease from him and succumbed in two to five days. Buboes were noted in only one case, in which the cervical glands were affected and melaena was present, in addition to the pneumonic signs. Shortly after death patches of post-mortem staining appeared in most instances. Yersin's serum was given therapeutically without success, and was also employed prophylactically. Isolation of the sick in a special hospital; 10 days quarantine of contacts; disinfection of dwellings, bedding, and clothing by formalin, creolin, or carbolic acid; the use of insect powder; and a rat campaign, were the preventive measures adopted. The attendants on the patients protected themselves effectually by wearing masks.

C. B.

Abdominal Plague.

Knowles (R.). **A Case of Plague.—Abdominal Type.**—*Indian Med. Gaz.* 1912. Dec. Vol. 47. No. 12. pp. 478-479.

A boy died of bubonic plague in Jhansi in December 1911, when it was raging in that city. His sister, aged 8, was seized with fever, vomiting, and diarrhoea a day later, to which she

succumbed in five days' time. At the post-mortem examination there was no bubo externally, but the mesenteric glands were enlarged and haemorrhagic. The spleen was engorged. "The whole of the small intestine—but particularly its caecal end showed longitudinal ulcers. The sites of these were visible from the peritoneal aspect owing to their haemorrhagic character. The ulcers were large, shallow, with soft swollen edges, haemorrhagic bases and situated in the long axis of the feet [sic]. The contents of the small intestine—some blood and much mucus—resembled red currant jelly." Bacilli which resembled *B. pestis* morphologically were found with other bacteria in the mesenteric glands and in the spleen.

[As there is no record of a cultural examination, the diagnosis of plague is doubtful.]

C. B.

Treatment.

Aumann. **Beobachtungen nach Salvarsaninjektion bei einem Fall von menschlicher Bubonenpest.** [Salvarsan in the Treatment of Plague.]—*Deut. Med. Wochenschr.* 1912. Nov. 14. Vol. 38. No. 46. pp. 2166-2168.

A case of plague was removed from a steamer and was treated in Hamburg. 0·3 gm. of salvarsan was administered intravenously. Death occurred eleven days later from a secondary staphylococcic infection. The remedy appeared to increase the agglutinins in the blood, but had no favourable influence on the course of the infection.

C. B.

Roehr (C. G.). **The Surgical Treatment of Pest Bubo. How to Reduce the High Death Rate.**—*New York Med. Jl.* 1912. Oct. 19. Vol. 96. No. 16. pp. 800-801.

It is suggested that 2 per cent. formalin should be injected into plague buboes. The author records no cases treated in this way, but he has adopted the method for tubercular glands. The introduction of the formalin causes intense pain which is followed by anaesthesia in the course of a few minutes.

C. B.

Serums and Vaccines.

Darling (S. T.). **Two Cases of Anaphylactic Serum Disease over Six Years after the Primary Injection of Horse-serum (Yersin's Anti-pest Serum).**—*Arch. Internal Med.* 1912. Nov. 15. Vol. 10. No. 5. pp. 440-444.

A pathologist received a prophylactic injection of 10 cc. of Yersin's anti-plague serum in the year 1905 after performing an autopsy on a case of plague. Six years and four months later another similar dose was administered for the same reason. On the sixth day severe symptoms came on, great depression, syncope, feeling of utter prostration, difficulty in swallowing, ashy pallor, imperceptible pulse, yawning and sighing, and general urticaria

which continued for a fortnight. It was some days before he lost his haggard look and muscular pains.

A negro who was employed as mortuary attendant developed similar symptoms eight days after a second injection of anti-pest serum, the first dose having been given six years and four months before; he recovered more rapidly than the former patient. Fourteen others to whom prophylactic injections were given on the same day remained free from all troubles, but they had not been treated with horse serum at any time previously.

C. B.

DE JONGE (G. W. Kiewiet). **Het oordeel van Dr. de Raadt over het Pestvaccin.** [Dr. Raadt's Opinion of Inoculation against Plague.]—*Geneesk. Tijdschr. v. Neder.-Indie.* 1912. Vol. 52. No. 5. pp. 517-525.

In No. 2 of the above named journal DE RAADT contributes a paper on the results of inoculation in the epidemic of plague which prevailed in Java in 1911. (See this *Bulletin*, No. 2, p. 62.) He gave the following table.

		Attacks of plague.	Incidence per cent.
Number of uninoculated ...	101,875	879	0·86
Number of inoculated with Dutch vaccine	47,131	125	0·26
Number of inoculated with Haffkine's vaccine	9,595	16	0·16

Notwithstanding these favourable figures, he wrote slightingly of the influence of inoculation on plague prevention, and of the greater success obtained with HAFFKINE'S vaccine than with the Dutch. De Jonge combats these views and states that it can be shown mathematically that the chances that the lessened incidence among the inoculated was causal and not accidental, are 18,000 billion to one.

C. B.

UNCLASSED.

SHIBAYAMA (G.). **Experiments on Prophylactic Inoculation against Experimental Plague Pneumonia in Guinea Pigs.**—*Far Eastern Assoc. Trop. Med.: Trans. Second Biennial Congress held at Hongkong*, 1912. pp. 130-147.

It is not easy to excite pneumonic plague in guinea-pigs. They possess a bunch of hair at the aperture of their nostrils which acts as a filter. Five of these animals, kept for 24 hours in the vicinity of a patient whose exhalations gave rise to a profuse growth of the *B. pestis* on agar plates when he coughed, remained healthy. Exposing them to sprays of *B. pestis* emulsions results in cervical buboes and septicaemia. When plague virus is introduced directly into the lung by inoculating through the shaven skin of the right breast of these animals, they succumb to pneumonic plague on the fourth day.

Nucleo-protein was obtained by treating plague agar cultures with 1 per cent. NaOH, precipitating with acetic acid, and

collecting and drying the precipitate. Guinea-pigs were injected with 0·5-1·2 mgm. of this substance, and ten to thirty days later their immunity was tested by intrapulmonary inoculation. They all died. Killed broth cultures of *B. pestis* were little better. Only one animal survived out of ten which had been vaccinated, and 12-50 days later were submitted to the test of lung inoculation. 18 mgm. of a killed agar culture protected 2 out of 10 guinea-pigs from the effects of intrapulmonary injection of the plague bacillus. Twenty out of thirty-one guinea-pigs survived lung inoculation given 12-47 days after they had been vaccinated with a living attenuated culture of the *B. pestis*. Ten guinea-pigs were vaccinated with killed agar plague emulsion, and nine days later with living avirulent plague bacilli. When their immunity was tested by lung inoculation 12-24 days afterwards, five survived. Twenty guinea-pigs were prepared in a similar manner, except that they received in addition injections of anti-pest serum; fifteen lived, their immunity being proved 11-73 days later.

The author gives this summary of his experiments:—

Killed plague bacilli may confer a certain degree of prophylactic power on animals, but not so high as the living attenuated bacilli. This fact has been affirmed by Kolle and Strong in their subcutaneous inoculation tests. Nucleo-protein seems to confer little immunity, if any. Inoculation with living attenuated bacilli may incur inoculation losses, and therefore one of the following methods is proposed in practice: to inoculate with the living attenuated plague bacilli after conferring some degree of immunity by inoculation with killed bacilli from agar cultures, or to inoculate with a mixture of the living avirulent strain and anti-plague serum.

The highest degree of immunity can be conferred by the last procedure.

C. B.

Brooks (Ralph St. John). **The Opsonic Index in Plague Vaccination.**—*Brit. Med. Jl.* 1912. Oct. 26. pp. 1098-1099.

Plague bacilli contain about 1/10 their weight of nucleo-protein. Groups of rats, ten in each, were inoculated with 1/10 mg. of plague bacilli, repeated 35 days later; with 1 mg. nucleo-protein; with 1/10; 1/100; 1/12,000 mg. nucleo-protein; with 1/10 mg. nucleo-protein hydrolysing for 8 months; and with 9/10 mg. washed bacilli respectively. The serum of each series was pooled and the opsonic index determined. The smallest response was elicited by the smallest dose of nucleo-protein, and the largest by the double dose of bacilli. The opsonic curve began to rise 10 hours after inoculation, and attained its maximum on the second to the fourth day. At the end of a week it fell, but had not quite sunk to its normal level in three weeks. The injection of washed bacilli and of hydrolysed nucleo-protein caused no elaboration of opsonins in rats. Nevertheless 1/10 mg. of hydrolysed nucleo-protein, which corresponds with that contained in 1 cc. of Haffkine's prophylactic, caused the appearance of opsonins in the serum of eleven persons into whom it was injected. In them the maximum response was observed later than in the rats. The curve was at its highest on the fifth and sixth days. The

response was somewhat greater in six who had been inoculated with HAFFKINE's vaccine on a previous occasion. The author's conclusions are as follows:—

"1. The substance which produces a rise in the opsonic index in immune pest serum is the nucleo-protein contained in the bodies of the bacilli.

"2. The washed bodies of the bacilli when used as a vaccine do not cause any increase in the opsonic index.

"3. Within limits, the larger the dose the greater the opsonic reponse.

"4. A repeated dose of vaccine raises the index above the maximum of the first inoculation, even after the curve has fallen to its normal level.

"5. In the early stages, in rats, the opsonic response and the degree of protection aroused rise together.

"6. In human beings the maximum response is observed to be much later than in the case of laboratory rats, and the response in a series of previously sensitised persons is somewhat greater than in a series of persons not previously vaccinated.

"7. The local and constitutional effects of nucleo-protein vaccination compare very favourably with those observed in other methods of protective inoculation."

C. B.

MANAUD (A.). **Les Facteurs Météorologiques et Climatologiques dans l'Etiologie de la Peste.**—*Revue d'Hyg. et de Police Sanit.* 1912. Nov. 20. No. 11. pp. 1125-1157.

A study of the seasonal prevalence of bubonic plague from the fourteenth century onwards shows that it is essentially a disease of summer and autumn in temperate climates. In support of this view the author quotes references to the bubonic plague outbreaks in Italy and France in the 14th century; to those in Milan in 1630; in Moscow in 1771; in London in 1578, 1593, 1613, 1603, 1625, 1636, and 1665; in Vienna in 1679; in Gratz in 1680; in Dantzig in 1709; in Moscow in 1711; in Marseilles in 1720; in Odessa in 1812; in Eastern Mongolia in 1898; in Oporto in 1899; and in San Francisco in 1907.

In hot climates bubonic plague prevails at that time of the year when the temperature ranges between 10° and 30° C. Temperatures below 10° C. and above 30° C. are inimical to its spread. In Bombay the epidemic is at its maximum in March, and at its minimum in July. In Egypt outbreaks of bubonic plague occur in the period November to June; the cases are most numerous in March or April. In the epidemics which took place in Aden in the years 1904 and 1905, and in that at Jedda in 1907, February and March were the months of maximum incidence. In Siam the author observed that February was the month of greatest prevalence in the outbreak of the year 1909, which comprised 191 attacks. Therefore the seasonal spread of bubonic plague is similar in all tropical climates, and it is not greatly influenced by the presence or absence of the rains.

It is a fact of familiar observation that rat-fleas are more numerous in the summer than in the winter in temperate climates. They become less lively, and their reproduction ceases in cold weather. Thus an explanation is afforded of the greater incidence of plague during the summer in temperate zones. In

tropical regions temperatures above 30° C. not only cause a reduction in the number of the fleas per rat, but are also hostile to the survival of *B. pestis* in the flea's stomach: hence bubonic epidemics subside during the hottest months in the tropics.

Pneumonic plague outbreaks arise in the winter; thus in the year 1348 pneumonic plague raged in Avignon from January to March; in Poland in January 1349, where it was followed by the bubonic form in the summer of that year; in the Tyrol, Hungary, Russia, Norway, and England in the winter months of the years 1348-1352; in Wetlianka, Astrachan, from October, 1878 to April, 1879; and in Manchuria during the winter 1910-1911. Insects are not concerned in the transmission of pneumonic plague. Infection is conveyed from man to man in droplets of saliva and sputum. [This paper forms a valuable contribution to the history of plague.]

C. B.

LIST OF REFERENCES.

Compiled by R. L. Sheppard, Librarian of the Bureau.

The references are to papers published since July 1, 1912, except in the case of the Kala Azar Section, where, to secure continuity with the lists in the *Kala Azar Bulletins*, some earlier references are given. Only titles which have been verified from the original papers are included. A few medical journals were not accessible, so that there are some omissions. Titles omitted from the present list will, as far as possible, be inserted in future lists. The Lists of References will be continued quarterly.

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See also **Dysentery (B. Unclassed).**

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